Intravenous Immunoglobulins Are a Therapeutic Option in the Treatment of Multiple Sclerosis Relapse

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**Objective:** The objective of the study is to evaluate the efficacy and tolerability of intravenous immunoglobulin (IVIG) monotherapy in the treatment of multiple sclerosis (MS) relapse.

**Background:** High-dose intravenous methylprednisolone (IVMP) and plasmapheresis have been shown to shorten the recovery period of an MS relapse. Options for those who have contraindications for or are unresponsive to these treatments are very limited. Intravenous immunoglobulin has been used experimentally in these situations, even though there are no previous studies on its efficacy as monotherapy in MS relapse.

**Subjects and Methods:** Twelve consecutive MS patients with acute MS relapse were treated with IVIG 0.4 g/kg per day for 5 days, and the next 5 patients received IVMP 1000 mg/d for 3 days. Volumetric brain magnetic resonance imaging (MRI) and clinical evaluation using expanded disability status scale (EDSS) were performed at baseline and at 3 weeks after treatment. EDSS score after 1 year of the treatment was collected from the patient records. MRI evaluation was performed blindly but not the clinical examination and EDSS scoring.

**Results:** A significant reduction in the volumes of T2-, fluid-attenuated inversion recovery-, and gadolinium-enhanced lesions was detected in the IVIG-treated group, but not in the IVMP-treated patients. The difference between the groups did not reach statistical significance. The EDSS score improved equally in both groups.

**Conclusions:** Intravenous immunoglobulin did not show inferiority compared with IVMP in the treatment of an acute MS relapse evaluated clinically and radiologically. Therefore, we suggest that IVIG may be tried as a therapy in acute MS relapse, especially in case of contraindications to IVMP and plasmapheresis.

**Key Words:** intravenous immunoglobulin, exacerbation of multiple sclerosis, EDSS, volumetric MRI

(Clin Neuropharm 2011;34: 84–89)

**M**ultiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system, which often exhibits a relapsing-remitting (RR) disease course. High-dose intravenous methylprednisolone (IVMP) is used as the standard treatment for MS relapse as it has been shown to increase the rate of recovery from acute relapse,\(^1,2\) improve the permeability of the blood-brain barrier (BBB), and suppress gadolinium (Gd) enhancement on magnetic resonance imaging (MRI) in acute demyelinating lesions.\(^3,4\) Although IVMP is mostly well tolerated, there are patients who experience significant adverse effects or derive no benefit from IVMP. Apart from IVMP, only plasmapheresis has proved effective in the treatment of MS relapse,\(^5,6\) so other treatment options are clearly called for. Intravenous immunoglobulins (IVIGs) are polyvalent human immunoglobulin G (IgG) preparations purified from large plasma pools obtained from thousands of healthy donors. They are indicated as the treatment of choice in primary immune deficiencies and in a number of inflammatory and autoimmune diseases.\(^7–14\) The immunomodulatory effects of IVIG have been attributed to a range of biologic functions of the polyvalent human IgG, which are mediated by either the Fc part or the antigen-binding F (ab\(^2\)) part of the IgG molecule.\(^10,14,15\) Owing to these effects, IVIG may be beneficial in the treatment of acute relapses of MS and therefore could offer a valuable alternative for those who have contraindications to IVMP or plasmapheresis or are unresponsive to these treatments. It has been shown that IVIG in combination with IVMP is not superior compared with IVMP alone in the treatment of acute MS relapse.\(^16,17\) Furthermore, IVIG did not improve a long-term visual function after treatment of acute optic neuritis (ON) compared with placebo,\(^18\) but its efficacy as monotherapy in MS relapse has not been previously studied.

In this study, we evaluated the efficacy of IVIG monotherapy in the treatment of MS relapse using volumetric cerebral MRI measurements and clinical evaluation including expanded disability status scale (EDSS).\(^19\) Owing to broad anti-inflammatory properties, we hypothesized that IVIG could prove beneficial in the treatment of acute MS relapse and therefore could offer an alternative for patients with this condition.

**SUBJECTS AND METHODS**

The study was conducted at the Department of Neurology in Tampere University Hospital, Tampere, Finland. It was approved by the ethics committee of the hospital, and all patients gave a written informed consent before study entry. Patients who had received IVMP in the preceding 8 weeks or immunosuppressive treatment in the preceding nine months were excluded. Characteristics of the patients are shown in Tables 1 and 2. Seventeen consecutive patients with acute MS relapse as rated by the criteria of McDonald et al\(^20\) were included. All patients had definite MS according to the criteria of McDonald et al.\(^20\)

Twelve consecutive patients received IVIG 0.4 g/kg per day (Endobulin; Baxter AG, Vienna, Austria) for 5 days. The control group included the next 5 patients who received standard treatment of IVMP 1000 mg/d for 3 days.

The study design was open-label except for MRI analyses, which were performed blindly from coded images by an experienced neuroradiologist (P.D.). The volumetric brain MRI and clinical evaluation were performed at baseline (at relapse immediately before treatment) and at 3 weeks after the first dose of study medication. The EDSS score at remission before the current relapse as well as the EDSS score 1 year after the

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DOI: 10.1097/WNF.0b013e31820a17f3
treatment was collected from the patient records. Examinations were performed by the same experienced neuroradiologist (P.D.) and neurologist (H.K.). Adverse events were evaluated during each day of the study drug infusion and at the final study visit at 3 weeks.

MRI Examinations Segmentation and Volumetric Analysis

All brain MRI examinations were performed using a 1.5-T MRI unit (Philips Gyroscan ACS NT 1.5T; Intera, Best, the Netherlands). The MRI protocol included a sagittal T1-weighted localizer, axial T1-weighted spin echo (SE) (echo time [TE] = 12 milliseconds, repetition time [TR] = 500 milliseconds, number of sequence averages [NSA] = 2, field of view [FOV] = 250 mm, matrix 256 × 256, slice thickness = 3 mm, slice gap = 0 mm, 46 slices), axial dual echo (T2/PD) sequence (TE = 24/10 milliseconds, TR = 3000 milliseconds, NSA = 1, turbo factor [TF] = 8, FOV = 250 mm, matrix 256 × 256, slice thickness = 3 mm, slice gap = 0 mm, 46 slices), axial fluid-attenuated inversion recovery (FLAIR) sequence (TE = 100 milliseconds, TR = 6000 milliseconds, inversion time = 2000 milliseconds, TF = 26, NSA = 2, FOV = 230, matrix = 228 × 256, slice thickness = 5 mm, slice gap 1 mm, 26 slices), and T1-weighted with magnetization transfer contrast sequence with and without contrast enhancement (TE = 13 milliseconds, TR = 550 milliseconds, NSA = 2, FOV = 230, matrix = 205 × 256, slice thickness = 5 mm, slice gap 1 mm, 26 slices). Computerized semiautomatic segmentation and volumetric analyses were carried out using Anatomatic™ (Tampere University/Tampere University Hospital, Tampere Finland)21 operating in a Windows environment. The interobserver and intraobserver variability in volumetric results has been reported elsewhere.21,22 The volumetric accuracy of the Anatomatic program was analyzed as previously described.22 Good head repositioning was controlled using the same head coil, the same anatomical locations, and the same pack of images in different MRI sequences.

The MRI outcomes analyzed were the number and volume of Gd-enhancing lesions, the volumes of T1, T2, and FLAIR lesions, as well as brain volume. T2-hyperintense plaques were analyzed from 3-dimensional (3D) T2 fast SE (FSE) images, T1-hypointense plaques from 3D T1 SE images, and FLAIR lesions from FLAIR images. Brain volumes for atrophy estimation were performed from T1 FSE images. Both T2 FSE and T1 SE were 3D in nature because the slice thickness was 3 mm, and the gap was 0 mm. Because the FLAIR sequence was not 3D in nature, the MS lesion volumes in the gap between 2 slices were estimated by multiplying the average cross-sectional area of the plaque structures by the gap thickness. The number and volume of enhancing lesions were documented from Gd-enhanced Tesla1–magnetization transfer contrast images. Gadolinium diethylenetriaminepentaacetic acid was administered intravenously as a bolus of 0.2 mmol/kg.

Statistical Analysis

Comparisons of measurements before and after treatment in the IVIG or IVMP groups were made using Wilcoxon signed rank test or paired t test. Between the 2 groups, statistical comparisons were carried out using Mann-Whitney U test for all measured parameters and changes (Δ) in measurements.

RESULTS

Effects of IVIG Therapy on Volumetric MRI Measurements

In the IVIG-treated group, an improvement in most MRI measurements was observed. The median volumes of T2 lesions decreased from 5.55 to 4.78 cm³ (P = 0.015) and of FLAIR lesions from 16.30 to 13.69 cm³ (P = 0.002). The median volumes of Gd-enhanced lesions decreased from 0.29 to 0.18 cm³ (P = 0.007) and their median number from 2.5 to 2.0 (P = 0.002). There were no significant changes in the volumes of T1 lesions or brain volumes. In the IVMP-treated group, the changes in pretreatment and posttreatment measurements were not statistically significant (Table 3). The differences of changes in MRI measurements between the IVIG group and the IVMP group did not reach statistical significance.

Effects on EDSS and Tolerability

After the 5-day course of IVIG therapy, the EDSS scores decreased from a mean of 3.8 to 2.6 (P = 0.002, Table 1). Likewise, the EDSS scores of the IVMP group improved from 4.7 to 3.9 (P < 0.05, Table 1). The differences between the groups were not significant. Comparison between the EDSS scores at 3 weeks and 1 year after treatments did not reveal significant changes in the studied groups (P > 0.05).

### Table 1. Clinical Characteristics of MS Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients Treated With IVIG (n = 12)</th>
<th>Patients Treated With IVMP (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SE, yrs</td>
<td>40.2 ± 3.0</td>
<td>34.4 ± 4.2</td>
</tr>
<tr>
<td>Sex, no. male/no. female</td>
<td>5/7</td>
<td>0/5</td>
</tr>
<tr>
<td>Duration of MS, mean ± SE, yrs</td>
<td>5.8 ± 0.9</td>
<td>5.2 ± 1.6</td>
</tr>
<tr>
<td>Time since previous relapse, mean ± SE, mo</td>
<td>17.6 ± 21.0</td>
<td>5.0 ± 3.2</td>
</tr>
<tr>
<td>EDSS1 score at remission, mean ± SE</td>
<td>2.4 ± 0.3†</td>
<td>3.8 ± 0.9</td>
</tr>
<tr>
<td>EDSS2 at acute relapse, mean ± SE</td>
<td>3.8 ± 0.3‡</td>
<td>4.7 ± 0.8§</td>
</tr>
<tr>
<td>EDSS3 3 wk after treatment, mean ± SE</td>
<td>2.6 ± 0.3‡</td>
<td>3.9 ± 0.9§</td>
</tr>
<tr>
<td>EDSS4 1 y after treatment, mean ± SE</td>
<td>3.0 ± 0.4‡</td>
<td>4.2 ± 0.8</td>
</tr>
</tbody>
</table>

*EDSS1 versus EDSS2, P = 0.002 comparison between pretreatment evaluation and immediately at relapse and posttreatment EDSS in the IVIG-treated group.
†EDSS2 versus EDSS3, P = 0.03 comparison between pretreatments and one-year posttreatment in the IVIG-treated group.
‡EDSS2 versus EDSS3, P = 0.002 comparison between evaluations immediately at relapse and posttreatment in the IVIG-treated group.
§EDSS 2 versus EDSS3, P = 0.04 comparison between evaluations immediately at relapse and posttreatment in the IVMP-treated group.
### TABLE 2. Clinical Data of the Individual Patients of the Study

<table>
<thead>
<tr>
<th>Patient/ Treatment</th>
<th>Sex</th>
<th>Age, y</th>
<th>Disease Duration, y</th>
<th>Baseline Treatment</th>
<th>Time Between Current and Previous Relapse, mo</th>
<th>EDSS at Remission</th>
<th>EDSS at Acute Relapse Before IVIG</th>
<th>EDSS 3 wk After Treatment</th>
<th>EDSS 1 y After Treatment</th>
<th>Symptom of the Relapse</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/IVIG</td>
<td>Male</td>
<td>52</td>
<td>6</td>
<td>Interferon B-1a IM</td>
<td>2</td>
<td>2.0</td>
<td>4.0</td>
<td>3.0</td>
<td>2.0</td>
<td>Spinal (partial paraparesis)</td>
</tr>
<tr>
<td>2/IVIG</td>
<td>Female</td>
<td>50</td>
<td>11</td>
<td>Interferon B-1b</td>
<td>UNK (years)</td>
<td>1.5</td>
<td>3.5</td>
<td>1.5</td>
<td>1.0</td>
<td>Cerebellar (ataxia of lower extremities)</td>
</tr>
<tr>
<td>3/IVIG</td>
<td>Female</td>
<td>37</td>
<td>0,7</td>
<td>Interferon B-1b</td>
<td>5</td>
<td>3.0</td>
<td>5.5</td>
<td>4.0</td>
<td>4.5</td>
<td>Spinal (partial paraparesis)</td>
</tr>
<tr>
<td>4/IVIG</td>
<td>Female</td>
<td>43</td>
<td>7</td>
<td>Interferon B-1b</td>
<td>34</td>
<td>3.0</td>
<td>5.0</td>
<td>3.0</td>
<td>3.0</td>
<td>Right hemiparesis</td>
</tr>
<tr>
<td>5/IVIG</td>
<td>Male</td>
<td>31</td>
<td>8</td>
<td>Glatiramer acetate</td>
<td>10</td>
<td>4.0</td>
<td>5.0</td>
<td>4.0</td>
<td>5.5</td>
<td>Right-leg weakness</td>
</tr>
<tr>
<td>6/IVIG</td>
<td>Female</td>
<td>29</td>
<td>2.5</td>
<td>None</td>
<td>3</td>
<td>0.0</td>
<td>2.0</td>
<td>1.0</td>
<td>1.0</td>
<td>Spinal (partial paraparesis)</td>
</tr>
<tr>
<td>7/IVIG</td>
<td>Male</td>
<td>31</td>
<td>3</td>
<td>Glatiramer acetate</td>
<td>5</td>
<td>2.0</td>
<td>3.0</td>
<td>2.5</td>
<td>2.5</td>
<td>Left-leg weakness</td>
</tr>
<tr>
<td>8/IVIG</td>
<td>Female</td>
<td>41</td>
<td>10</td>
<td>Glatiramer acetate</td>
<td>3</td>
<td>2.5</td>
<td>3.0</td>
<td>2.5</td>
<td>2.5</td>
<td>Right-leg weakness</td>
</tr>
<tr>
<td>9/IVIG</td>
<td>Female</td>
<td>37</td>
<td>9</td>
<td>Interferon B-1a 44 µg SC</td>
<td>7</td>
<td>3.0</td>
<td>4.0</td>
<td>3.0</td>
<td>4.0</td>
<td>Right-eye ON</td>
</tr>
<tr>
<td>10/IVIG</td>
<td>Male</td>
<td>28</td>
<td>3</td>
<td>Interferon B-1b</td>
<td>27</td>
<td>2.5</td>
<td>3.0</td>
<td>2.5</td>
<td>2.5</td>
<td>Spinal (partial paraparesis)</td>
</tr>
<tr>
<td>11/IVIG</td>
<td>Female</td>
<td>41</td>
<td>4</td>
<td>Interferon B-1a 44 µg SC</td>
<td>9</td>
<td>3.0</td>
<td>5.0</td>
<td>2.0</td>
<td>UNK</td>
<td>Right hemiparesis</td>
</tr>
<tr>
<td>12/IVIG</td>
<td>Female</td>
<td>63</td>
<td>6</td>
<td>Interferon B-1a IM</td>
<td>65</td>
<td>2.5</td>
<td>3.0</td>
<td>2.5</td>
<td>4.5</td>
<td>Left-eye ON</td>
</tr>
<tr>
<td>1/IVMP</td>
<td>Female</td>
<td>46</td>
<td>6</td>
<td>Interferon B-1b</td>
<td>3</td>
<td>6.0</td>
<td>6.5</td>
<td>6.0</td>
<td>6.0</td>
<td>Brain stem and cerebellar (ataxia, dysarthria, nystagmus)</td>
</tr>
<tr>
<td>2/IVMP</td>
<td>Female</td>
<td>43</td>
<td>11</td>
<td>Interferon B-1b</td>
<td>11</td>
<td>3.0</td>
<td>4.5</td>
<td>3.5</td>
<td>3.5</td>
<td>Right-eye ON</td>
</tr>
<tr>
<td>3/IVMP</td>
<td>Female</td>
<td>25</td>
<td>3</td>
<td>Interferon B-1b</td>
<td>5</td>
<td>2.0</td>
<td>3.0</td>
<td>2.0</td>
<td>2.0</td>
<td>Left hemiparesis</td>
</tr>
<tr>
<td>4/IVMP</td>
<td>Female</td>
<td>28</td>
<td>2</td>
<td>None</td>
<td>2</td>
<td>6.0</td>
<td>6.5</td>
<td>6.0</td>
<td>6.0</td>
<td>Cerebellar (ataxia)</td>
</tr>
<tr>
<td>5/IVMP</td>
<td>Female</td>
<td>30</td>
<td>4</td>
<td>Betaseron (interferon B-1b) + mitoxantrone</td>
<td>5</td>
<td>2.0</td>
<td>3.0</td>
<td>2.0</td>
<td>3.5</td>
<td>Cerebellar (ataxia)</td>
</tr>
</tbody>
</table>

IM indicates intramuscular; ON, optic neuritis; SC, subcutaneous; UNK, unknown.
In the IVIG group, 3 of 12 patients experienced headache, and 1 patient had a skin infection at the site of the intravenous cannula. None of the adverse events were serious. In the IVMP group, the only adverse event was an elevation in blood glucose in a diabetic subject.

**DISCUSSION**

Because the efficacy of IVMP in the treatment of acute MS relapses has been well shown,1–4 this therapy is recommended as the first-line treatment in acute relapses. Also, plasmapheresis has been shown to exert a beneficial effect, but it is a relatively demanding procedure.5,6 However, not all patients are responsive to IVMP, and there are various contraindications to this therapy, for example, severe diabetes or history of marked psychiatric disorders.

A recent review on the management of MS relapse states that IVIG may be considered for the treatment of relapses in patients who are unresponsive to steroids or have a contraindication to their use.23 To our understanding, this recommendation is based mostly on clinical experience, because previous studies on IVIG monotherapy in the treatment of MS relapses are lacking. The efficacy of IVIG in combination with IVMP has been analyzed in 2 studies that did not demonstrate the superiority of such combination over IVMP alone.10,17 In a study by Roed et al.,19 IVIG or placebo was administered to treat ON. The study population comprised patients with acute ON, but not necessarily MS. The primary end point was the visual function 6 months after ON. There was no difference between the groups at 6 months, but the short-term effect was not evaluated in this study.28 On the other hand, a recent study reported that IVIG might have beneficial effects in patients with insufficient recovery from ON, if treatment with high-dose IVMP fails.24,25

Our study was originally designed to identify a set of genes induced by IVIG during MS relapse; these data have been reported recently elsewhere.26 In the present clinical part of the study, we detected an equal effect on EDSS scores in both IVIG and IVMP groups. However, a significant reduction in the volumes of T2-, FLAIR-, and Gd-enhanced lesions as well as in the number of Gd-enhanced lesions over a period of 3 weeks after treatment was found in the MRI analyses from IVIG group only. In the IVMP group, MRI findings remained without significant changes. The lack of significant differences in the MRI measurements between the groups may be related to a relatively small number of patients involved in the study. Also, a more active disease in the IVMP-treated group as suggested by shorter disease duration and a shorter period between current and previous relapses may be another explanatory factor. In the IVIG group, the time from the previous relapse was 17.6 months and in the IVMP group, 5.0 months.

The effects of monthly IVIG treatments on relapse rate and MRI activity in MS have been studied by 4 randomized double-blind studies27–29 that all demonstrated reduction of the annual relapse rate by IVIG. A significant beneficial effect on EDSS28 and decrease in the number of Gd lesions in brain MRI were also demonstrated. However, the Prevention of Relapses With IVIG trial failed to support earlier observations of a beneficial effect of IVIG in RRMS.30 Recently, IVIG has been shown to decrease the relapse risk postpartum and is, in fact, the only immunomodulatory drug for MS that can be administered during lactation.31–33 Based on these studies, the main indication for the use of IVIG in MS is to reduce relapses during pregnancy or breast-feeding when other therapies may not be used safely.34,35 Although recent studies suggest the efficacy of IVIG in clinically isolated syndromes,36 additional evidence is needed to make any recommendations for the use of IVIG in this condition. In studies on chronic progressive MS, a reduction in brain atrophy was found in secondary progressive MS,38 and a borderline significant delay in time to sustained progression on EDSS was found in patients with primary progressive MS.39 These 2 studies suggest that IVIG might exert neuroprotective effects in advanced MS with pronounced neurodegenerative changes.

Previous studies have not demonstrated an effect of IVIG on BBB permeability,40 although the combination of IVIG and IVMP has been shown to reduce Gd enhancement in brain MRI.15 In our study, the decreases in the volume and the number of Gd-enhanced lesions and in the volume of FLAIR lesions indicate that IVIG exerts a beneficial effect in the acute inflammatory stage of lesion development, which is mostly associated with increased BBB permeability. This is consistent with the observations from the genetic part of our study, strongly suggesting that the regulation of cell proliferation, in particular, the regulation of T-cell proliferation, is a mechanism of action of IVIG.41 Modulation of humoral and cell-mediated immune responses including effector mechanisms of T cells, macrophage functions, production of cytokines, and inhibition of the complement system has been reported also by others.41 An effect on T2-leSION volume is consistent with a reduction in the overall extent of macroscopic tissue damage. The absence of changes in T1-leSION volume or brain volume indicates that a 5-day course of IVIG is not sufficient to influence axonal degeneration over a 3-week period. Despite improvement in the EDSS scores in the IVMP-treated group, no significant changes were seen on MRI over the 3-week period. Because the improvement in BBB by

**TABLE 3. MRI Measurements Before and After Treatment of Relapse, Median (Minimum-Maximum) (in cm³)**

<table>
<thead>
<tr>
<th></th>
<th>Before IVIG</th>
<th>After IVIG</th>
<th>Before IVMP</th>
<th>After IVMP</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 lesion volume</td>
<td>1.06 (0.12–6.85)</td>
<td>0.93 (0.10–7.31)</td>
<td>1.09 (0–3.28)</td>
<td>0.76 (0–5.24)</td>
</tr>
<tr>
<td>T2 lesion volume</td>
<td>5.55 (0.32–12.21)</td>
<td>4.78 (0.33–10.97)</td>
<td>8.54 (0.82–25.28)</td>
<td>7.19 (0.73–25.91)</td>
</tr>
<tr>
<td>FLAIR lesion volume</td>
<td>16.30 (5.62–27.77)</td>
<td>13.69 (5.13–24.36)</td>
<td>31.18 (2.12–51.29)</td>
<td>27.88 (1.5–46.35)</td>
</tr>
<tr>
<td>Gd lesion number</td>
<td>2.0 (0–6)§</td>
<td>2.0 (0–8)</td>
<td>1.0 (0–8)</td>
<td></td>
</tr>
<tr>
<td>Gd lesion volume</td>
<td>0.29 (0.85–85)§</td>
<td>0.18 (0–79)§</td>
<td>0.29 (0–2.29)</td>
<td>0.29 (0–2.3)</td>
</tr>
<tr>
<td>Brain volume</td>
<td>1111.97 (860.03–1342.62)</td>
<td>1109.63 (866.55–1342.66)</td>
<td>1067.64 (896.45–1230.46)</td>
<td>1050.45 (882.54–1225.78)</td>
</tr>
</tbody>
</table>

*P = 0.015, comparison between pretreatment and posttreatment measurements in the IVIG-treated group.
†P = 0.002, comparison between pretreatment and posttreatment measurements in the IVIG-treated group.
‡P = 0.002, comparison between pretreatment and posttreatment measurements in the IVIG-treated group.
§P = 0.007, comparison between pretreatment and posttreatment measurements in the IVIG-treated group.
IVMP has clearly been documented elsewhere,3,4 our observation suggests that the improvement in BBB permeability attained by IVMP is transient and may not be maintained up to 3 weeks. Intravenous immunoglobulin used at a dose of 0.4 g/kg body weight is a standard treatment for several autoimmune disorders. Previous studies have shown that monthly IVIG treatment in doses ranging from 0.15 to 2 g/kg body weight attenuates clinical and MRI disease activity in patients with RRMS.27–30 The optimal dose of IVIG treatment in MS, however, has yet to be established, although 1 study suggested 0.2 g/kg once monthly as being as efficacious as 0.4 g/kg in reducing the relapse rate and clinical disability in RRMS.39 The present results indicate that a dose 0.4 g/kg is well tolerated and safe in the treatment of MS relapse.

We found IVIG monotherapy to be as efficacious as the current drug of choice (IVMP) for treatment of acute MS relapse evaluated both clinically and using various volumetric MRI parameters. Therefore, we suggest that IVIG may be tried as a therapy for patients in MS exacerbation, especially in case of contraindications to IVMP and plasmapheresis. Because this study had a relatively low number of patients, the data should be validated in larger placebo-controlled study.

REFERENCES


